Equivalent electronic circuit model of cardiovascular system

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Abstract

Diastolic and systolic properties of all four cardiac chambers were simulated by using equivalent electronic circuits. Two models were made: the *linear model* with linear diastolic and systolic pressure-volume relationships, and the *nonlinear model*, which is a closer approximation to situation *in vivo*, with nonlinear diastolic pressure-volume relationship. Models of four cardiac chambers were connected to models of pulmonary and systemic circulation, pace-making system of the heart, and pericardium. The result was a functioning model of the cardiovascular system. Aortic pressure, pulmonary artery pressure, atrial pressures, cardiac output, and ventricular volumes were analysed in three different settings of the model, where either atrial contractions or pericardium were excluded, were analysed and graphically represented in time.

1 Introduction

The human heart consists of *four cardiac chambers*, namely a pair of atria and a pair of ventricles. All four of them are surrounded by *pericardium. Heart valves* placed between atria and ventricles, and between ventricles and great arteries determine the direction of blood flow. Ventricles are the main pumps which eject blood into the pulmonary and systemic circulation. Each beat of the heart, called the cardiac cycle can be divided into diastole and systole; during diastole the ventricles are being passively filled with blood, whereas in systole they actively eject it.

2 Methods

Equivalent electronic circuit of cardiovascular system was made by using Electronics Workbench 5.0c computer programme under Windows operating system. The electrical variables in the electronic circuit are the so-called *equivalent variables* of those in the cardiovascular system [1-4]: Voltage, current, charge, resistance and capacitance in the electronic circuit are equivalent to blood pressure, flow, volume, resistance, and capacitance in the cardiovascular system. Ground potential is equivalent to atmospheric pressure. Abbreviations used in the text:

A	Parameter constant
AoP	Aortic pressure
AP	Atrial pace-making sub-circuit
Batt	Battery
C	Capacitance
Cap	Capacitor
CO	Cardiac output
D	Diode
EDV	End diastolic volume
E_{max}	Maximal elastance during single contraction
G	Ground
k	Parameter constant
LA	Sub-circuit of the left atrium
LAtP	Left atrial pressure
LV	Sub-circuit of the left ventricle
M	Multiplier
MPC	Model of pulmonary circulation
MSC	Model of systemic circulation
NDS	Nonlinear dependent source
Opamp	Operational amplifier
P	Pressure
PAP	Pulmonary artery pressure
P_{max}	Maximal pressure developed during isovolumic contraction
R	Resistor
RA	Sub-circuit of the right atrium
RAtP	Right atrial pressure
RV	Sub-circuit of left ventricle
TWVS	Three way voltage summer
V	Volume
V_0	Unstressed volume
VCS	Voltage controlled switch
V_D	Intercept on the volume axis in pressure-volume diagram
VLV	Volume of the left ventricle
VP	Ventricular pace-making sub-circuit
VRV	Volume of the right ventricle

3 Results

3.1 Linear model

Linear model of a heart chamber is a more understandable and valid version of the model that was used in previous simulations [1-4]. It has linear diastolic and systolic pressure-volume relationships.

3.1.1 Diastolic properties

The linear model has a constant capacitance, which results in linear diastolic pressure volume relationship. Diastolic properties are described by eqn (1). Mode of simulation is presented in Fig. 1.

$$V = C \cdot P + V_0 \tag{1}$$

3.1.2 Systolic properties

During diastole heart chamber is filled with blood. Its end diastolic volume (EDV) depends on its capacitance, pressure at the end of diastole and its unstressed volume (eqn (1)). With larger EDV a heart chamber produces higher pressure during isovolumic contraction according to the Law of Starling. Within the physiologic range of EDV there is a linear relation between EDV and maximum pressure developed during isovolumic contraction [5, 6]:

$$P_{\max} = E_{\max}(EDV - V_D) \tag{2}$$

Mode of simulation of the Law of Starling by using electronic circuits is presented in Fig. 1.

3.2 Nonlinear model

The nonlinear model differs from the linear model in its diastolic properties. Complex expressions were developed to describe the nonlinear diastolic pressure-volume relationship of cardiac chambers [7], but we used a simpler exponential expression that was used in previous mathematical simulations of cardiovascular physiology [8, 9].

To simulate this nonlinear relationship some other electronic components were added to the linear model circuit (Fig. 1). They measure the voltage (P_1) at one end of the capacitor and apply a function of that voltage (P_2) to the other end of the capacitor, so that in the end the voltage difference (ΔP) on the capacitor follows eqn (3):

$$\Delta P = \frac{1}{k \cdot C} \cdot \ln\left(\frac{P_1 + A}{A}\right) + \frac{V_0}{C} \tag{3}$$

Mode of simulation is presented in Fig. 1.

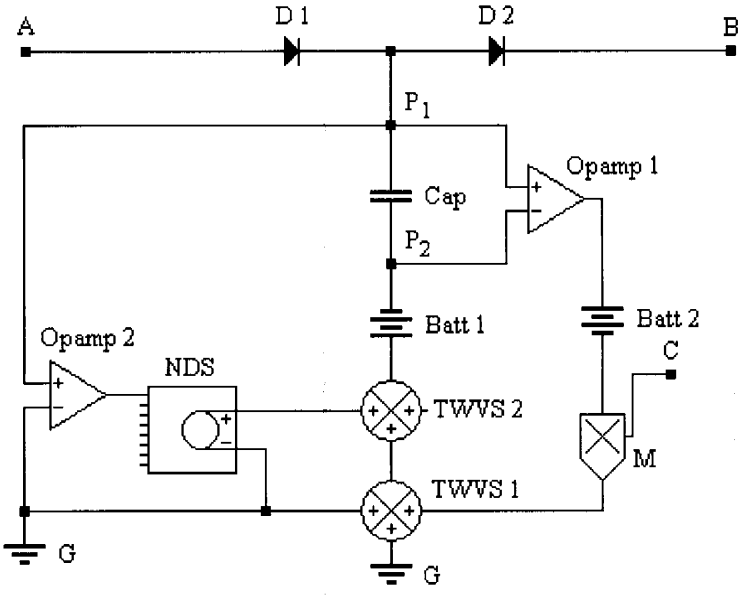

Figure 1: The linear model circuit (components surrounded by the grey line). *Diastolic properties*: Cap simulates the heart chamber with constant capacitance. Negative voltage applied to the capacitor by Batt 1 sustains baseline voltage difference on the capacitor, which is equivalent to the unstressed volume of the heart chamber. *Systolic properties*: Opamp 1 measures voltage difference on Cap and multiplies it by the value its capacitance. The result is voltage equivalent to the volume of the heart chamber. Batt 2 subtracts voltage equivalent to volume intercept. M multiplies its input with the value of E_{max} solely in systole when output from the pace-making system (not shown in the illustration) connected to C exceeds zero. D1 and D2 are diodes simulating heart valves, A and B are connectors to the circulation.
The nonlinear model (the entire circuit). *Diastolic properties*: Opamp 2 measures voltage inside the cardiac chamber (P_1) and applies it to NDS. This element transforms the input according to eqn (3) and applies the output through TWVS 2 and Batt 1 to the other end of the capacitor (P_2). *Systolic properties*: same as in linear model.

3.3 Simulation of the complete cardiovascular system

The model of the complete cardiovascular system includes pulmonary and systemic circulation, four cardiac chambers, pericardium and the pace-making sub-circuits of the atria and ventricles (Fig. 2).

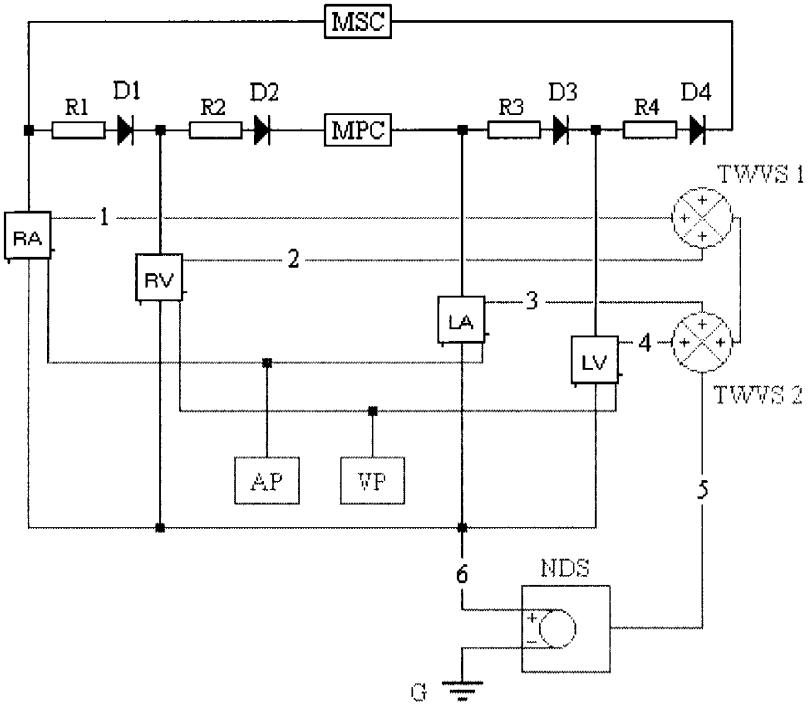

Figure 2: Scheme of the complete cardiovascular system. The current (blood flow) circuit, drawn in black colour, consists of sub-circuits of pulmonary and systemic circulations (MSC and MPC) and the four cardiac chambers (RA, RV, LA, and LV). D1, D2, D3 and D4 are equivalent to tricuspid, pulmonary, mitral and aortic valve, respectively. Resistors (R1, R2, R3, and R4) simulate the resistance of heart valves. Sub-circuits and components drawn in grey colour determine heart rate and successive contractions of atria and ventricles (AP and VP), and simulate the pericardium.
Simulation of the pericardium: voltages equivalent to volumes of the four cardiac chambers (1, 2, 3, and 4) are summed by TWVS 1 and TWVS 2. The sum (5) serves as input to NDS. This component transforms its input, which is equivalent to total heart volume, into voltage equivalent to intra-pericardial pressure according to eqn (4) and applies it equally to all four cardiac chambers (6).

Systemic and pulmonary circulations are modelled as a sequence of resistors and capacitors, similarly as in previous electronic simulations of cardiovascular system [1-4].

Heart chambers are modelled according to the nonlinear model. Parameter values defining each chamber's diastolic (A, k, V_0) and systolic (E_{max} and V_D) properties are identical to those used by Amoore and Santamore [8, 9] in their mathematical model of cardiovascular system.

Pericardium is simulated as an external pressure applied equally to all cardiac chambers. Volume of the pericardium (V_p) is the sum of volumes of all four cardiac chambers. Intra-pericardial pressure (P_p) is a function of pericardial volume and follows eqn (4), where Vp_0 is unstressed volume of pericardium at which transmural pressure is zero.

$$P_P = A \cdot \left(e^{(V_p - V_{p0}) \cdot k} - 1 \right) \tag{4}$$

The pace-making system of the heart is simulated by two separate sub-circuits: VP sub-circuit and AP sub-circuit, which determine ventricular and atrial contractions, respectively. This separation is based on the fact that atrial contraction precedes ventricular contraction.

The element central to both VP and AP is the clock, which acts as a pace-maker (Fig. 3). The VP clock is set to: heart rate: 1Hz, amplitude: 1V, duty cycle: 20%, whereas AP clock is set to the same heart rate and amplitude, but a duty cycle of 85%. The output of the clock is connected to two voltage controlled switches. They regulate the filling and emptying of the capacitor, which is equivalent to contraction and relaxation of a heart chamber. In systole, determined by the rise of VP clock output to value of 1 V, capacitor is filled by a battery through resistor R1. The time constant of contraction is determined by C and R2 [3, 4]. When the VP clock output voltage falls to zero, the capacitor empties through R2. The time constant of contraction is determined by C and R2.

AP sub-circuit is similar to VP sub-circuit, yet slightly different. In this sub-circuit the fall of the AP clock output dictates systole of the atria, therefore the voltage controlled switches have inverse characteristics as those in VP sub-circuit. As already noted this difference enables successive contraction of atria and ventricles.

As a demonstration, selected variables (AoP, PAP, RAtP, LAtP, CO, VRV, and VLV) were analysed and graphically represented in time in three different settings of the model described above (Fig. 4).

At the beginning of the analysis both pericardium and AP sub-circuit were disconnected (Fig. 4A) from the model, therefore only ventricles were contracting. Afterwards the AP sub-circuit was reconnected (Fig. 4B), which means that the atria were also contracting. AoP, PAP, and CO slightly increased, atrial pressures became double-waved (arrow in Fig.4B); the first wave is due to atrial contraction, the second wave is due to filling of the atria during ventricular systole. Ventricular volumes also slightly increased mainly because of end diastolic filling of ventricles, again due to atrial contractions. Finally, the

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pericardial pressure was applied to all four cardiac chambers (Fig. 4C). This resulted in dramatic fall of AoP, PAP, and CO. Atrial pressures remained double-waved, but there was reduced difference between RAtP and LAtP, due to reduced LAtP and increased RAtP. End diastolic volumes of both ventricles were significantly decreased, whereas end systolic volumes decreased only slightly.

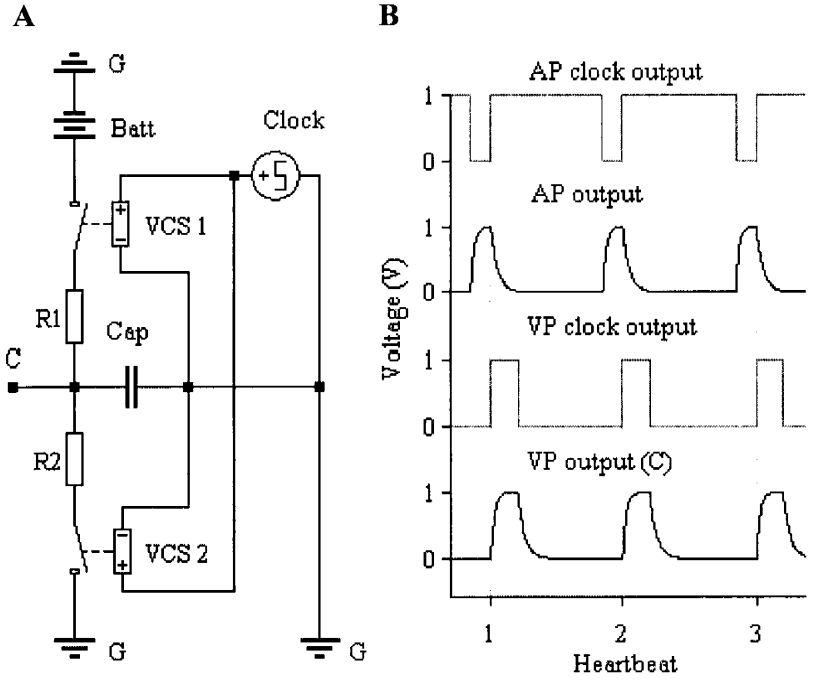

Figure 3: A: Ventricular pace-making (VP) sub-circuit. The output of the clock is connected to two VCS1 and VCS2. These switches regulate filling and emptying of the Cap, which is equivalent to contraction and relaxation of the ventricles. In ventricular systole, dictated by the rise of VP clock output voltage to value of 1V, VCS1 is closed while VCS2 is open. Therefore the Cap is charged with voltage of 1V by Batt through resistor R1. When the VP clock output voltage falls to zero, VCS1 opens, while VCS2 closes. The capacitor empties through R2 to the ground (G). The output of VP sub-circuit (C) is connected to the multiplier in sub-circuits simulating right and left ventricle (Fig. 1).
B: Output values of AP clock, AP sub-circuit, VP clock and VP sub-circuit. Note that atrial contraction precedes ventricular contraction.

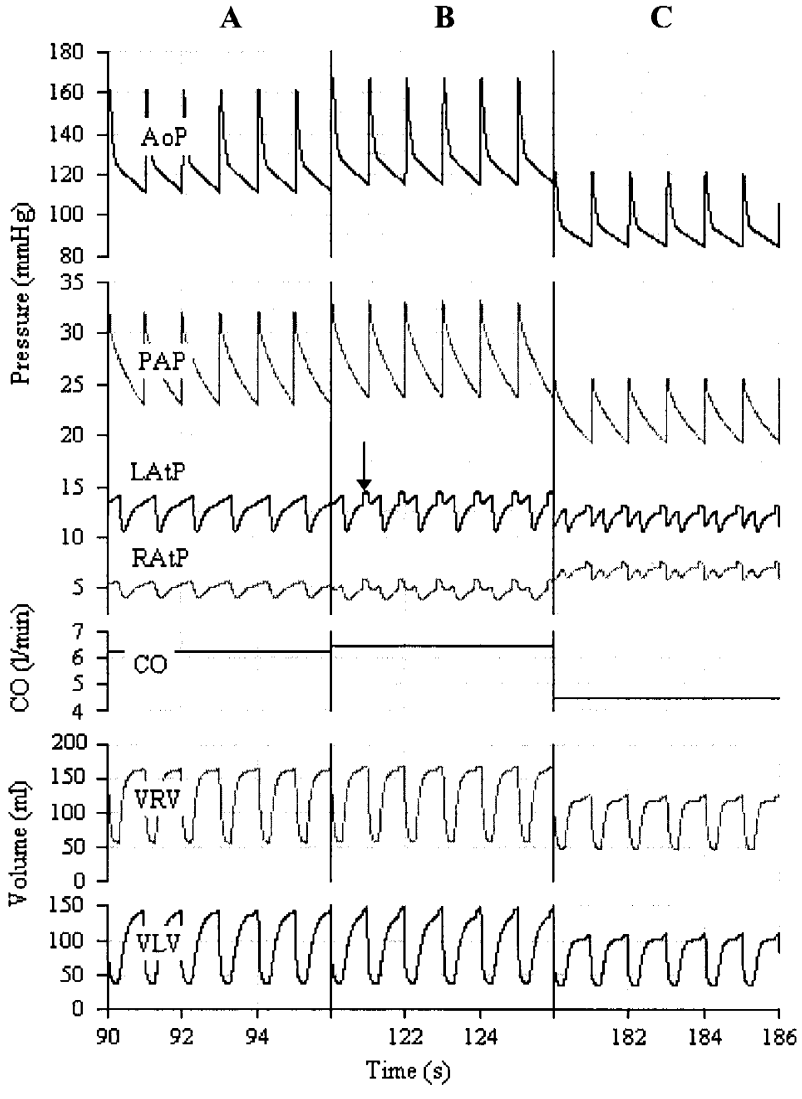

Figure 4: Analysis of selected variables in simulation of the complete cardiovascular system. From top to bottom: AoP, PAP, LAtP, RAtP, CO, VRV, and VLV.
A: model of cardiovascular system without pericardium; atria are not contracting.
B: without pericardium; atria are contracting. Note slightly increased Aop and PAP, double-waved atrial pressures (arrow), increased CO, and end diastolic spikes in VRV and VLV due to atrial contractions.
C: includes pericardium and contracting atria. Note the fall of arterial pressures, decreased difference between RAtP and LAtP, decreased CO, and the restriction of both ventricular volumes.

4 Discussion

It was shown, that nonlinear diastolic properties of cardiac chambers can be simulated by using equivalent electronic circuits. This way greater similarity to conditions *in vivo*, described by mathematical expressions, is combined with advantages of Electronics Workbench computer programme: analysis of selected variables is shown as a graph, parameters defining properties of the heart or the circulation can be changed easily, programme is commercially available and runs on personal computer, very little mathematical knowledge is needed to use the programme or the model of cardiovascular system.

Using the present equivalent electronic circuit, four-chambered heart has been simulated successfully. The time course of atrial pressures and ventricular volumes is different that in two-chambered heart model [1-4]. Double-waved RAtP is very similar to *in vivo* jugular venous pulse, which reflects changes in RAtP [10]. End diastolic filling of the ventricles, caused by atrial contractions, is also observed *in vivo*.

Pericardium surrounds all four cardiac chambers and represents a significant restriction to diastolic filling [11]. Applying intra-pericardial pressure to cardiac chambers caused a decrease of ventricular end diastolic volumes (atrial volumes were not measured), which further resulted in the fall of AoP, PAP, and CO. However, restrictive role of pericardium may prevent excessive distension of cardiac chambers in conditions with increased atrial pressures (congestive heart failure). Furthermore, reduced difference between LAtP and RAtP was observed. With pericardium included, excessive filling of one cardiac chamber can influence, indirectly through increased intra-pericardial pressure, filling of other cardiac chambers. A similar phenomenon is observed *in vivo* in cardiac tamponade, where additional volume inside pericardial sac increases intra-pericardial pressure and in extreme cases causes equalisation of atrial pressures.

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